EDITORIAL

The Athlete's heart and the endless pursuit of prediction factors

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The Athlete's heart is nowadays a well-known condition, consequence of continuous sport practice and is considered as a kind of structural and physiological adaptation to extreme training. Since the first reports, concern about its potential risks replaced the idea that elite competitors are the healthiest individuals capable of such a high performance.

It was not until recently that dilatation, hypertrophy and enhanced parasympathetic tone characteristics of exercise-induced cardiac changes were acknowledged as possible risk factors for development of atrial fibrillation[1], creating a connection between athlete's heart concept and the arrhythmogenic process. In fact, recent works have demonstrated that the risk of AF is significantly higher in athletes compared with non athletes[2], challenging cardiologists and electrophysiologists to unravel this major issue: how to predict the incidence of potential severe arrhythmia, responsible for thromboembolic events, poor quality of life and impaired left ventricular function in a young and otherwise healthy population?

The paper by Hasdemir *et al.*[3] intends to address this fundamental question. By studying cardiac electrical and structural properties in young and old football players, authors were able to demonstrate the correlation between novel electrocardiographic parameters such as filtered P wave dispersion and aging – a traditional established prediction factor for AF. Indeed, a previous work[4] has already demonstrated the role of P wave duration and P wave dispersion on predicting idiopathic AF in normal individuals, stating that those are simple and useful electrocardiographic markers.

It is intriguing, however, that Karakaya *et al.*[5] showed, after including 50 athletes and 40 sedentary controls, that ECG measurement of P wave duration and dispersion did not differ significantly in both groups and could not be used as reliable predictors for AF developed in trained athletes.

Echocardiographic assessment of left atrial diameter is another simple tool recognized as a possible marker for AF[6]. Grismo *et al.*[6] presented a follow-up of 149 healthy long-term trained skiers, concluding that left atrial enlargement was a marker associated with AF incidence. Again, this work represents an effort to reach prediction with feasible and widely accessible methods and it is rather plausible that long term physical activities may induce chamber dilatation and fibrosis that ultimately create a favorable substrate for the disease. Nonetheless, in this issue of the journal, Hasdemir *et al.*[3] proved the influence of aging on atrial electrophysiology to be independent of left atrial diameter, questioning its role as a risk factor. In fact, a recently published study that describes the structural changes induced by exercise in elite competitors suggests that atrial remodeling is often present in athletes without AF[7].

At this point, all authors face a major problem: it is not always easy trying to address complex questions with simple answers. Since 1910, when Lewis in London and, Rothberger and Winterberg in Vienna established atrial fibrillation as a clinical condition[8], it has been proved to be a complex multifactorial entity. Accordingly, it could take also complex methods to make possible detection of most susceptible individuals. What causes some athletes but not others to develop AF is still unknown.

Maybe, with enhanced evaluation tools, more valuable risk markers could be achieved. Detailed analysis of P wave morphology, including accurate onset and offset points, is difficult with 12-lead ECG. Such analysis is possible with the high resolution of Signal-averaged ECG, another eligible noninvasive predictor of AF that has not yet been widely tested in exercise practitioners.

A number of genetic loci and genes related to the disorder have been reported[9]. The discovery of genes underlying AF offers another attractive way of providing insight into the mechanisms responsible for this arrhythmia and possibly new risk factors. Technological improvements in DNA analysis could enable researchers to implement these data into a personalized, genomic-oriented medicine.

Whether electrocardiografic, echocardiografic or other kind of marker do have useful prediction power remains to be elucidated. New data suggesting that P wave analysis can be considered as a reliable predictor of AF in exercise practitioners need to be confirmed by larger case-control studies. Meanwhile elite exercise competitors should be carefully followed and evaluated to rule out arrhythmias.

Once reliable and clinically efficacious predictors of AF were achieved, a fundamental question will still remain: will they help physicians finding a threshold limit for intensity and duration of physical activity that prevents AF without limiting cardiovascular benefits[10], such as lower aortic stiffness and enhanced endothelium-dependent dilatation?

References

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